Amendments to the Specification:

Please replace the paragraph on page 3 lines 14-22 with the following amended paragraph:

Figures 1 (SEQ ID No. 1 and 2), 2 (SEQ ID No. 3 and 4), and 3 (SEQ ID No. 5 and 6) show the nucleotide and amino acid sequence of the preferred tumor associated uPAR deletion variants del4, del4+5, and del5. The sequence of deletion variant del5 according to Figure 3 (SEQ ID No. 5 and 6) is already described in a public database (Genbank Accession No. U8839). However, no hint towards a tumor selective expression of this deletion variant can be found there. Figures 4A, 4B (middle strand: overlap SEQ ID No. 9), 5A, 5B (middle strand: overlap SEQ ID No. 10), and 6A, 6B (middle strand: overlap SEQ ID No. 11), show a comparison of the nucleotide and amino acid sequence of the deletion variants with the corresponding wild-type sequence (SEQ ID No. 7 and 8).

Please replace the paragraph on page 12 lines 6-7 with the following amended paragraph:

Figure 1 shows the nucleotide and amino acid sequence for the uPAR deletion variant del4 (nucleotide: SEQ ID No. 1 and amino acid: SEQ ID No. 2).

Please replace the paragraph on page 12 lines 9-10 with the following amended paragraph:

Figure 2 shows the nucleotide and amino acid sequence for the uPAR deletion variant del4+5 (nucleotide: SEQ ID No. 3 and amino acid: SEQ ID No. 4).

Please replace the paragraph on page 12 lines 12-13 with the following amended paragraph:

Figure 3 shows the nucleotide and amino acid sequence for the uPAR deletion variant del5 (nucleotide: SEQ ID No. 5 and amino acid: SEQ ID No. 6).

Please replace the paragraph on page 12 lines 15-17 with the following amended paragraph:

Figure 4 shows a comparison between the nucleotide sequence (A: SEQ ID No. 1) and the amino acid sequence (B: SEQ ID No. 2) and the wild-type sequence (nucleotide: SEQ ID No. 7 and amino acid: SEQ ID No. 8) for the deletion variant del4.

Please replace the paragraph on page 12 lines 19-21 with the following amended paragraph:

Figure 5 shows a comparison between the nucleotide sequence (A: SEQ ID No. 3) and the amino acid sequence (B: SEQ ID No. 4) and wild-type sequence (nucleotide: SEQ ID No. 7 and amino acid: SEQ ID No. 8) for the deletion variant del4+5.

Please replace the paragraph on page 12 lines 23-25 with the following amended paragraph:

Figure 6 shows a comparison between the nucleotide sequence (A: SEQ ID No. 5) and the amino acid sequence (B: SEQ ID No. 6) and the wild-type sequence (nucleotide: SEQ ID No. 7 and amino acid: SEQ ID No. 8) for the deletion variant del5.

Please replace the paragraph on page 13 lines 12-13 with the following amended paragraph:

Figure 10 shows peptides, which were selected for immunization. Identical amino acids are accentuated in yellow (uPAR del4+5: SEQ ID No. 12; uPAR del4: SEQ ID No. 13; uPAR del5: SEQ ID No. 14).

Please replace the paragraph on page 13 lines 27-30 with the following amended paragraph:

Figure 15 shows various uPAR expression plasmids. In addition to the protein sequences described, all uPAR plasmids encode the native N-terminally located signal sequence of uPAR (22 amino acids) which is post-translationally clipped off during export (pRcRSV-SuPAR-DI: SEQ ID No. 15; pRcRSV-GPI-uPAR-DII+IIICP and pRcRSV-GPI-uPAR-DII+III: SEQ ID No. 16).

Please replace the paragraph on page 23 lines 2-21 with the following amended paragraph:

Qualitative RT-PCR for uPAR

Cellular mRNA was isolated using the Micro Fast Track™ kit (Invitrogen, Karlsruhe, Germany) and cDNA synthesized using the cDNA-Cycle™ kit (Invitrogen, Karlsruhe, Germany). PCR was performed in a master mix containing 1 x PCR buffer (PE/Applied Biosystems, Forster City, CA), 2 mM of each desoxyribonucleotide triphoshate (Stratagene, LaJolla, CA), 2 μM of uPAR-specific primers for uPAR-exons 1 and 6 (SEQ ID No. 17: forward: CTCCACACCTGCGTCCCA, SEQ ID No. 18: reverse: CTTGCAGCTGTAACACTG; Metabion, Martinsried, Germany) (Fig. 16), 1.0 U

AmpliTaq Gold^{TM} DNA polymerase (PE/Applied Biosystems), and 2 µl cDNA in a total volume of 50 µl. The amplification profile consisted of denaturation at 94 °C for 1 min, annealing at 49 °C for 45 sec, and primer extension at 72 °C for 45 sec in a 30-cycle reaction. Twenty µl of the PCR products were separated electrophoretically in 4-12 % TBE-PAGE (Novex), stained with SYBR Green (Biozym Diagnostica GmbH, Hess. Oldendorf, Germany), and analyzed with the Fluor-S MultiImager (BioRad, München, Germany) using the Multi-Analyst PC software (BioRad, München, Germany). As an internal control, glyceraldehyde-3-phosphate dehydrogenase (GAPDH) was co-amplified. Amplified cDNA was separated by agarose gel electrophoresis (2%) and visible PCR products were sequenced.

Please replace the paragraph on page 24 lines 12-28 with the following amended paragraph:

For amplification of both uPAR cDNA types (mRNA encompassing exons 2, 3, and 4 [del2+3+4 or uPAR-2/3/4]; cDNA encompassing exons 2, 3, and 6 and lacking exon 4 and 5 [del4+5 or uPAR-4/5]), separate PCR reactions applying the same forward primer targeted to exon 2 (5'-GACCTCTGCTGCAGGACCACGAT-3': SEQ ID No. 19), but different reverse primers were used. For the del2+3+4 cDNA, a primer targeted to exon 4 (AGGTAACGGCTCCGGGAAT: SEQ ID No. 20) was designed to obtain a fragment length shorter than 200 bp (Fig. 16), which allows efficient PCR performance on the LC. Using this primer set, fragments either from the uPAR-wt and/or from the del5 variants are amplified. For del4+5 cDNA, a primer overlapping the expected exon 3-6 boundary (5'-TTTCAAGCTCCAGGACAGAGTT-3': SEQ ID No. 21) was used. In both assays identical hybridization probes (uPAR-fluorescein-labeled 5'-GGTACAGCTTTTCTCCACCAG-CTCCA-3': SEQ ID No. 22) and uPAR-LC640 5'-CTCTTCTCCTTCTTCCCACAAGCG-3': SEQ ID No. 23) located in exon 3 were used. Amplification of GAPDH was performed using the sense primer (5'-

TGGTCACCAGGGCTGCTTTTA-3': SEQ ID No. 24) and antisense primer (5'-TCCTGGAAGATG-GTGATGGGATTT-3': SEQ ID No. 25).

Please replace the paragraph on pages 27 line 4 to 28 line 4 with the following amended paragraph:

Example 2.2: Detection of uPAR antigen in CHO cells transfected with plasmids encoding uPAR variants

To elucidate whether alternatively spliced uPAR mRNA may be translated and posttranslationally processed, expression plasmids encoding wt-uPAR (DI+II+III) and various uPAR variants including uPAR-DII+III, del5 and del4+5 in a GPI-linked form were generated and stably transfected into CHO cells (Fig. 15). By ELISA, flow cytofluorometry, and Western blot analysis, synthesis and secretion of wt-uPAR and the uPAR variants were demonstrated in these cells. When lysates of the transfected cells were analyzed by the use of the Imubind uPAR-ELISA (American Diagnostica Inc.), a increased absorbance in samples derived from CHO-DII+III, CHO-del5 as well as CHO-del4+5 cells compared to the vector control was observed, which corresponds to a certain uPAR content concerning the standard curve of the test (Fig. 17). For flow cytofluorometric analysis, two different mAbs against uPAR were used, which are directed to different epitopes on DI (mAb IIIF10) and on DII/III (mAb HD13.1) domains of uPAR, respectively (Fig. 18A). With both mAbs, a distinct cell surface-associated reaction was observed with CHO cells transfected with GPI-linked uPAR-wt, the del5 as well as del4+5 plasmid variants (but not with the vector control), strongly indicating that the GPI-linked splice variants are presented at the cell surface. The DI-specific mAb IIIF10 (mapped epitope aa 52-60), as expected, did not bind to CHO-DII+III cells, which emphasizes the specificity of the analysis. In line with this, the DII-specific mAb IID7 but not the mAb IIIF10 reacted with lysates of CHO cells transfected with GPI-uPAR-DII+III plasmids (with and without the chemotactic peptide ^{88}SRSRY^{92} (SEQ ID No. 16); Fig. 15) in Western blot analysis (Fig. 18B an C). Whereas both mAbs bound to lysates of GPI-del5 (deletion of aa 136-180) transfected CHO cells, the DII-specific mAb IID7 (mapped epitope aa 125-132) did not react with the lysates of CHO-del4+5 cells transfected with an uPAR plasmid lacking the DII-encoding exons (deletion of aa 82-180). The rather broad bands in Western blot analysis as well as the increase of the apparent molecular weight of the various uPAR variants strongly indicate that these proteins - similarly as wt-uPAR – are glycosylated in CHO cells (Fig. 18B and C).